Available online at www.sciencedirect.com
SCIENCE DIRECT®
Clinical Biomechanics 21 (2006) 545–553
CLINICAL BIOMECHANICS
www.elsevier.com/locate/clinbiomech

Suprascapular nerve block disrupts the normal pattern of scapular kinematics

Sean P. McCully [a], David N. Suprak [b], Peter Kosek [c], Andrew R. Karduna [b,*]

[a] *Department of Anatomy, BioSciences Institute, University College Cork School of Medicine, Cork, Ireland*
[b] *Department of Human Physiology, University of Oregon, Eugene, OR 97403, USA*
[c] *Pain Consultants of Oregon, Eugene, OR 97401, USA*

Received 9 August 2005; accepted 1 February 2006

Abstract

Background. Patients with full thickness rotator cuff tears typically demonstrate an increase in scapular motion, both in the clinic and under controlled laboratory conditions. To better understand the mechanisms behind this pattern of motion, we propose a suprascapular nerve block as an appropriate model of dysfunction of the supraspinatus and infraspinatus, which are the two tendons most commonly affected in cuff tear patients.

Methods. Healthy subjects underwent testing for 3D scapular kinematics with a Polhemus magnetic tracking device and isometric force measurements during external rotation. A suprascapular nerve block was then performed with the injection of lidocaine into the suprascapular notch of each subject. Scapular kinematics and isometric force measurements were repeated after confirmation of the block.

Findings. The nerve block resulted in no significant changes in clavicular rotations and scapular posterior tilting. However, there was a significant increase in scapular external rotation and upward rotation. While kinematic changes returned to baseline within 25 min of the block, force measurements did not return to baseline until 75 min post-block.

Interpretation. The results of this study, especially those for upward rotation, are in general agreement with what has been found for patients with rotator cuff tears. While the supraspinatus and infraspinatus do not directly control the movement of the scapula, they appear to result in a compensatory change in scapular motion. Although more work needs to be done, it appears that abnormal scapular motion patterns observed in patients with cuff tears may therefore be compensatory in nature.
© 2006 Elsevier Ltd. All rights reserved.

Keywords: Shoulder; Rotator cuff tears; Scapular kinematics; Nerve block

1. Introduction

In the United States, it is estimated that the incidence of rotator cuff tears in patients over 40 years of age may be as high as 40%, making it one of the most prevalent complications associated with the adult shoulder (Matsen et al., 2004; Soslowsky et al., 1997). Although the etiology of rotator cuff tears is multifactorial, it has been suggested that if the synchronous pattern of motion between the scapula and humerus is disrupted, the rotator cuff tendons might become impinged under the coracoacromial arch (Fu et al., 1991). This may in part be due to the fact that alterations in scapular orientation can affect the amount of clearance in the subacromial space, as demonstrated in vivo with Magnetic Resonance Imaging (MRI) (Solem-Bertoft et al., 1993) and in cadavera models (Karduna et al., 2005; Wong et al., 2003).

In the clinic, alterations of scapular movement patterns are associated with several conditions that can accompany rotator cuff tears, such as muscle weakness (Nicholson, 1989), fatigue (Cohen and Williams, 1998), and paralysis (Matsen and Arntz, 1990). Several research studies have

* Corresponding author.
E-mail address: karduna@uoregon.edu (A.R. Karduna).

0268-0033/$ - see front matter © 2006 Elsevier Ltd. All rights reserved.
doi:10.1016/j.clinbiomech.2006.02.001

quantitatively demonstrated that patients with cuff tears have increased scapular motion when compared to healthy controls (Yamaguchi et al., 2000; Paletta et al., 1997; Deutsch et al., 1996; Mell et al., 2005). Since it is not practical to observe patients before they develop cuff tears, we do not know whether these abnormal patterns are causal or compensatory in nature. In this situation, controlled models are often useful in understanding an underlying pathology. To date, there has been extensive work in the development of animal (Soslowsky et al., 1996; Dejardin et al., 2001; Tillander et al., 2001) and cadavera (Parsons et al., 2002; Halder et al., 2002; Thompson et al., 1996) models of rotator cuff tears. However, an in vivo human model would provide additional information.

The majority of full thickness tears start with the supraspinatus tendon and then progress posteriorly to the infraspinatus tendon (Matsen et al., 2004; Sher, 1999). Therefore, interventions that result in dysfunction of these muscles are candidate models. One possibility is to selectively fatigue these muscles. Although we have had success with this approach for the infraspinatus in our laboratory (Tsai et al., 2003), the supraspinatus is more of a challenge. Another approach would be the use of a pharmacological nerve block. The suprascapular nerve branches from the superior trunk of the brachial plexus, and after passing inferiorly and laterally, goes deep to the trapezius muscle. The nerve then passes through the suprascapular notch and innervates both the supraspinatus and infraspinatus muscles (Pratt, 1991). Suprascapular nerve blocks are commonly performed clinically for pain relief of the shoulder due to conditions such as adhesive capsulitis and nerve entrapment (Tan et al., 2002; Shanahan et al., 2003; Karatas and Meray, 2002). However, several investigators have taken advantage of its innervation to perform nerve block studies for biomechanical evaluations of strength (Kuhlman et al., 1992; Howell et al., 1986; Colachis and Strohm, 1971) and kinematics (Howell and Kraft, 1991).

We propose the use of a suprascapular nerve block as an appropriate model of dysfunction of the supraspinatus and infraspinatus muscles. Specifically, the aim of this project is to examine the effect of a suprascapular nerve block on scapular kinematics. We hypothesize that this block will result in a compensatory increase in scapular rotations and decrease in glenohumeral motion.

2. Methods

2.1. Subjects

Fifteen subjects participated in this study (age range 20–33 years). There were seven females and eight males, with a mean age of 26 (SD 4) years, a mean height of 174 (SD 9) cm and a mean mass of 70 (SD 10) kg. Subjects were excluded from the study if they had any of the following: (1) less than 135° of active humeral elevation in the scapular plane; (2) prior shoulder surgery; (3) shoulder injury in the past six months; (4) presence of shoulder pain preventing the correct execution of tests; (5) allergies to lidocaine. Additionally, all subjects indicated that they had no history of cervical or shoulder pain or pathology and were recruited from a diverse university population. The dominant shoulder of each subject was tested. Approval for this study was obtained from the Institutional Review Board of the University of Oregon. All subjects were informed of the nature and details of the study and gave written and verbal consent prior to their participation.

2.2. Kinematic measurements

A Polhemus 3Space Fastrak (Colchester, VT, USA) was used for collecting three-dimensional in vivo kinematics of the shoulder complex. Subjects were asked to sit with their thoracic spine, scapula, and humerus exposed for receiver placement. Women were asked to wear a sports bra, which allowed access to the entire scapular region. Subject's sat with their feet flat on the floor at a comfortable width apart, back against the chair, and eyes fixed forward. This seated position was maintained throughout marker placement while a standing position was used for digitization and collection procedures.

The Fastrak is a magnetic tracking device that consists of a global positioning transmitter, three receivers (thoracic, scapular, and humerus), and a digitizing probe, which are hardwired to the system electronics unit. The transmitter was firmly attached to a vertical stand at a distance of approximately 50 cm behind the subject and was leveled. The first receiver was placed on the spinous process of the third thoracic vertebra using Spirit Gum adhesive and Micropore tape to fix the receiver to the skin. The second receiver was secured to a custom cuff made of Polyform (Sammons Preston Rolyan, Bolingbrook, IL, USA) splinting material, positioned on the distal humerus. This receiver was aligned between the medial and lateral epicondyles of the dominant arm and was secured with a Velcro strap. The final receiver was positioned over the scapula after mounting it on a custom made and previously validated scapular-tracking device machined from plastic. The base of the scapular-tracker is plastic and has a hinge joint that conforms to the spine of the scapula. From this base, an adjustable arm extends and contacts the acromion. The base and the arm contacting the acromion were attached to the skin with adhesive-backed Velcro strips placed above and below the spine of the scapula and on the flat part of the acromion just proximal to the origin of the deltoid muscle (Fig. 1). We have previously validated this technique for assessing scapular kinematics with an in vivo bone pin study (Karduna et al., 2001).

A series of standardized embedded axes were established. These axis systems were derived from a series of anatomical landmarks proposed by the shoulder sub-committee of the International Society of Biomechanics committee for standardization and terminology (Wu et al., 2005). The landmarks on each bony segment were digitized in the following order: seventh cervical vertebra, eighth

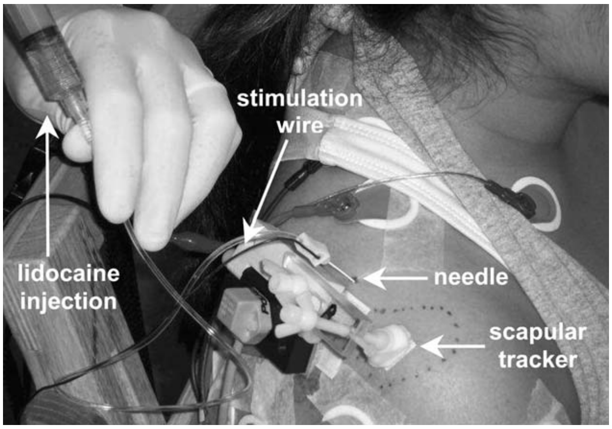

Fig. 1. Photograph of nerve block procedure demonstrating drug injection, stimulation wire, needle and scapular-tracker. The EMG electrodes were used for a separate study.

thoracic vertebra, sternal notch, xiphoid process, acromioclavicular joint, root of the scapular spine, inferior angle, posterio-lateral edge of acromion, medial epicondyle, lateral epicondyle, and center of humeral head. All landmarks were on the surface of each subject and could be digitized directly, with the exception of the humeral head. The center of the humeral head was determined as the point that moved the least with regard to the scapula when the humerus was moved throughout short arcs of mid-range glenohumeral motion and was calculated using a least squares algorithm (Harryman et al., 1990).

Standard matrix transformation methods were applied to determine the rotational matrix of the humerus with respect to the thorax or scapula and the scapula with respect to the thorax. Humeral rotations were represented using a standard Euler angle sequence where the first rotation defined the plane of elevation, second defined the amount of elevation, and the last rotation represented the amount of internal and external rotation (An et al., 1991). Scapular rotations were also represented by an Euler angle sequence of external rotation (retraction), upward (lateral) rotation and posterior tilting (Karduna et al., 2000). Two clavicular rotations, protraction/retraction and elevation/depression were used to describe scapular position with respect to the thorax (Karduna et al., 2001). Clavicular angles were derived from the location of the sternal notch and acromioclavicular joint, which were tracked with the thoracic and scapular receivers, respectively.

2.3. Maximum voluntary isometric force measurements

In order to best isolate the torque due to the supraspinatus and infraspinatus muscles, shoulder external rotation force was measured with a 3390-50, 50 kg compression load cell (Lebow, Troy, MI, USA). The load cell was connected to a mobile horizontal brace attached to the wall which could be adjusted and fixed for subject variations in height. Subjects were seated in a chair position that was marked for repeated placement. The load cell was adjusted so that the dorsal side of the subject's hand was in contact with a foam pad attached to the load cell when the arm was at the side, in neutral shoulder rotation with the elbow at 90° of flexion. A large weight was placed next to the front leg of the chair to prevent chair rotation during collection. The investigator also applied force to the subject's thigh to oppose rotational motion created from the force of contraction. The investigator positioned the subject's arm until a tone sounded, at which time the subject was instructed to produce a maximal shoulder external rotation torque for 3 s. The average force produced during the middle one second period of each maximal voluntary isometric contraction (MVC) was averaged and recorded. External rotation force data was used to confirm a successful nerve block, with an acceptance threshold of at least a 50% reduction in external rotation force from baseline (Colachis and Strohm, 1971; Kuhlman et al., 1992).

2.4. Testing protocol

Prior to data collection, the following warm-up procedure was performed: hanging humeral circumduction (15 clockwise/15 counter-clockwise rotations), horizontal shoulder flexion/extension, and shoulder abduction/adduction with a 2.3 kg mass in the dominant hand. The glenohumeral joint was then preconditioned by placing it in 90° of elevation in the coronal plane while the investigator passively rotated the shoulder. Subjects verbally confirmed when a good stretch was felt in the shoulder capsule and the investigator held the position for 15 s. Three internal and three external stretches were used to complete shoulder preconditioning.

Subjects were asked to stand while performing normal shoulder elevations in the scapular plane prior to and following the nerve block procedure. Scapular plane orientation was defined as approximately 30–35° anterior to the coronal plane. To ensure proper placement, real time confirmation of the position of the humerus was checked through on screen visual feedback from the magnetic tracking device. Prior to each collection the investigator positioned the arm in the correct scapular plane, moved the arm through 30° of humeral elevation/depression, and returned the arm to the subject's side. Shoulder elevation trials were collected with subjects standing at a marked position, eyes facing forward, elbow in full extension, with the thumb pointing toward the ceiling. The range of motion produced was subject dependant, but all trials began with the arm at the subject's side.

Two shoulder elevation trials were collected prior to a suprascapular nerve block. To control the velocity of motion, audible counts of 4 s were used during both shoulder elevation and depression (8 s total). Three shoulder elevations and depressions constituted one complete trial. Nine shoulder elevation trials were collected following the suprascapular nerve block. Immediately following each completed trial, MVC measurements as described above were collected. There was a 5 min rest period following the end of force collection and the beginning of the next kinematic trial.

2.5. Suprascapular nerve block

One of the authors (PK), who is a Board Certified Anesthesiologist, performed the nerve block procedure. Subjects were seated, with their head allowed to flex forward. The area around the shoulder was sterilized with betadine and the scapular spine was palpated bilaterally for comparison and accuracy. One inch above the junction of the middle and outer third of the scapular spine, the suprascapular nerve was targeted at the scapular notch using an insulated Stimuplex (STIMD2250/30) 22 GA × 2 in. 30° Bevel needle (B. Braun Medical Inc., Bethlehem, PA, USA) through a skin wheel of 0.2 ml of 1% lidocaine (Fig. 1). Nerve stimulation was achieved with a current of 0.6 mA and then reduced to confirm stimulation at 0.2 mA with needle repositioning as necessary using a Stimuplex-Dig 'Nerve stimulator for plexus anesthesia' (B. Braun Medical Inc., Bethlehem, USA). After aspiration did not result in blood, lidocaine 1.5% 1 ml was injected. In one subject, the notch was not identified, but stimulation confirmed proximity to the suprascapular nerve. In all cases, stimulation was abolished at 0.5 mA 30 seconds after the lidocaine dose. The remaining 5.7 ml of 1.5% lidocaine (total 100 mg) were injected and the needle was removed. A time stamp was recorded, and a countdown timer initiated, the moment the needle was withdrawn. Five minutes post-needle withdrawal, a manual muscle test at 15° of bilateral humeral abduction was performed to check for muscle weakness. Ten minutes following needle withdrawal, subjects were asked to stand, and the first of nine nerve blocked shoulder elevation trials commenced.

2.6. Data reduction and analysis

The percent reduction in external rotation force was calculated between the data collected immediately prior to and immediately after the nerve block. For each trial, scapular rotations were interpolated in 10° increments of humerothoracic elevation and averaged over the three elevations. Statistical tests were performed at humeral elevation angles from 20° to 120°, as this was the common range achieved by all subjects under all conditions. Kinematic and force data from the two trials conducted prior to the nerve block were used to assess reliability using the Intraclass Correlation Coefficient, ICC (3,1), and the Standard Error of the Measurement (SEM) (Portney and Watkins, 2000). For each dependent variable (scapular posterior tilting, upward rotation and external rotation, clavicular plane and elevation and glenohumeral elevation) a two way repeated measures analysis of variance (ANOVA) was performed with two within subject factors (elevation angle and block). If there was a significant effect of the block and a significant interaction between the two factors, follow-up paired *t*-tests were run at each humeral elevation angle. As an additional check of the effects of the nerve block, the maximum absolute change in rotation for each dependent variable was recorded for each trial and averaged over all of the subjects. The means of these were compared to zero with a one sample *t*-test. The alpha level was set at 0.05 for all analyses.

3. Results

Out of the original 15 subjects tested, 10 were included for the purposes of data analysis. Four subjects did not meet the 50% reduction in external rotation torque and one subject was so affected by the block that she could not elevate her arm without assistance.

The ICC values were 0.85 or better for all kinematic variables, except for clavicular plane values below 80° of humeral elevation. The SEM was 2° or less for all dependent variables, except for external rotation (Table 1). For external rotation force, the ICC was 0.92 and the standard error of the measurement was 0.8 kg.

There was no significant effect of the nerve block on posterior tilting, clavicular protraction and clavicular elevation. For scapular upward (lateral) rotation, there was a significant effect of the block ($P = 0.009$) and a significant interaction between elevation angle and block ($P < 0.001$). Follow-up *t*-tests indicated that the amount of upward rotation was significantly increased due to the block at humerothoracic elevations from 20° to 90° ($P < 0.01$ for all except 90°, where $P = 0.025$) (Fig. 2B). For scapular external rotation (retraction), there was a significant effect of the block ($P = 0.036$), but there was no significant interaction between humerothoracic elevation angle and block ($P = 0.916$). Follow-up *t*-tests indicated that the amount of external rotation significantly increased due to the block at humerothoracic elevations from 70° to 120° ($P < 0.05$) (Fig. 2C). For glenohumeral elevation, there was a significant effect of the block ($P = 0.021$) and a significant interaction between humerothoracic elevation and block ($P < 0.001$). Follow-up paired *t*-tests reveal that glenohumeral elevation was significantly decreased due to the block at humerothoracic elevations from 30° to 90° ($P < 0.05$) (Fig. 2F). When considering the maximum abso-

Table 1
Reliability of kinematic variables

	Posterior tilting	Upward rotation	External rotation	Clavicular plane	Clavicular elevation	Glenohumeral elevation
ICC range	0.95–0.98	0.97–0.99	0.86–0.95	0.71–0.93	0.97–0.99	0.85–0.99
SEM range [deg]	0.4–1.1	0.6–1.0	1.8–2.4	1.3–1.7	0.5–0.8	0.7–1.5

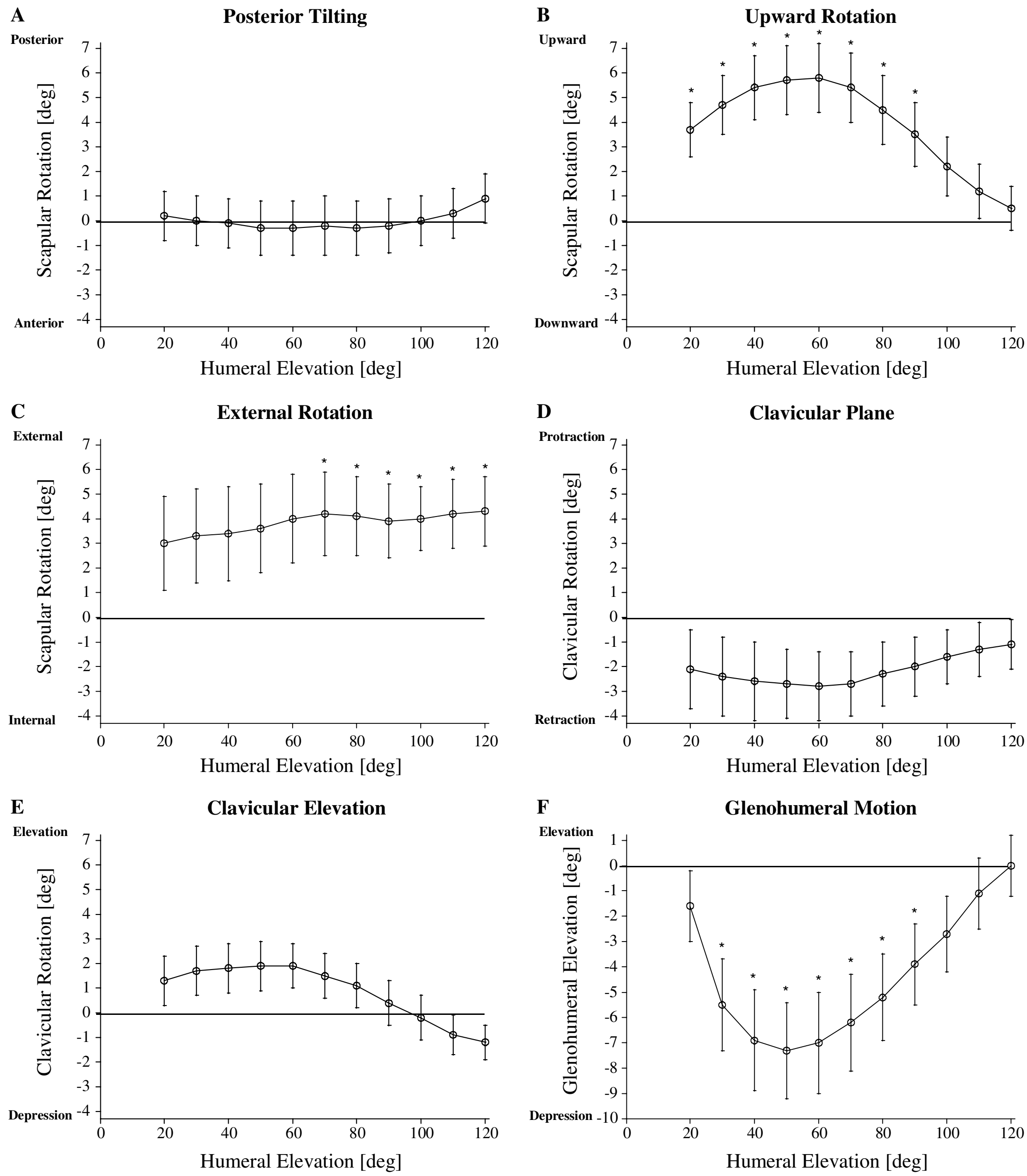

Fig. 2. Mean (SEM) differences between post- and pre-nerve block trials for (A) scapular posterior tilting, (B) scapular upward rotation, (C) scapular external rotation, (D) clavicular plane, (E) clavicular elevation and (F) glenohumeral elevation. $^{*}P < 0.05$.

lute change in rotations, all dependent variables demonstrated a significant change ($P < 0.001$) due to the block (Fig. 3).

The nerve block resulted in a mean external rotation force level which was 25% of the original baseline measurement. With subsequent trials, there was a linear force recovery, which was still significantly lower than baseline until the last trial (approximately 75 min after the block) (Fig. 4A). As an indicator of kinematic recovery, we looked at the changes in upward rotation at 60° of elevation, since that was where the maximum post-block changes were observed. Unlike force recovery, the changes in kinematics demonstrated a dramatic drop over the first several trials and were not significantly different from baseline by the third post-block trial (approximately 25 min after the block) (Fig. 4B).

4. Discussion

The results of the current study support our original hypothesis that a suprascapular nerve block results in a compensatory increase in scapular rotations and decrease in glenohumeral motion. Interestingly, when analyzing mean data, these changes were only observed for upward rotation and external rotation. However, when the absolute magnitude of the changes was analyzed, all six kinematic variables demonstrated significant changes. This was due to the fact that for some subjects there was an

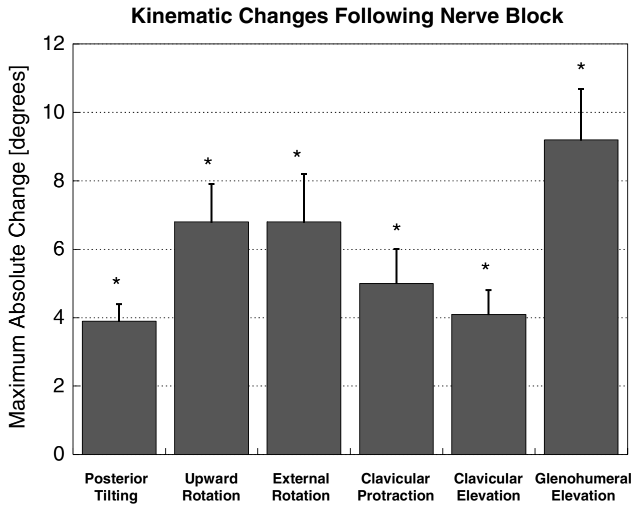

Fig. 3. Maximum absolute mean (SEM) changes between post- and pre-nerve block data.

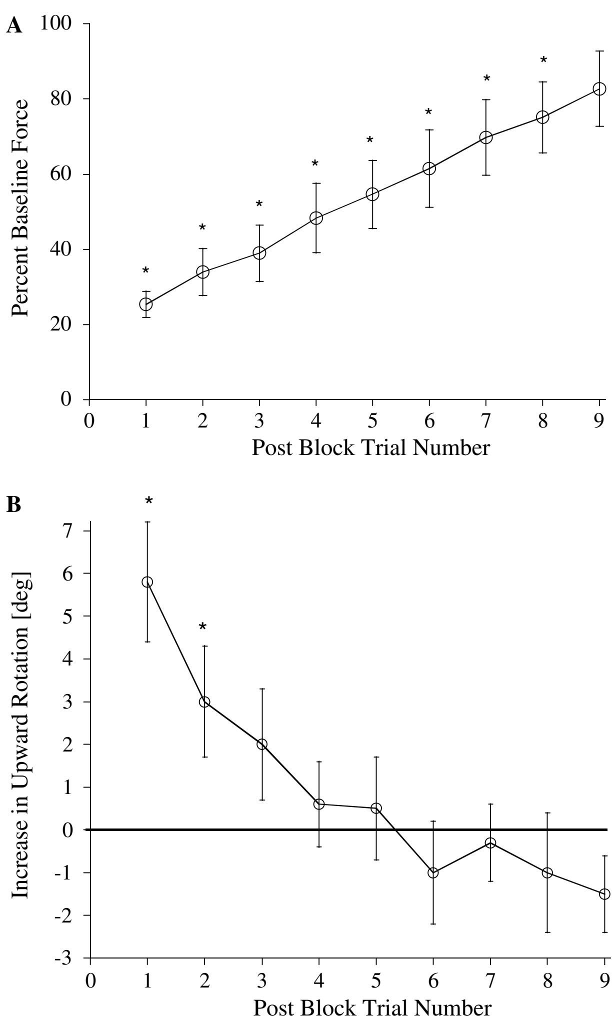

Fig. 4. Mean (SEM) values for (A) percent baseline external rotation force and (B) changes in upward rotation at 60° of elevation vs post-block trial numbers. $^{*}P < 0.05$.

increase in motion and for others there was a decrease in motion. Despite the fact that the muscles innervated by the suprascapular nerve (supraspinatus and infraspinatus) do not directly control the movement of the scapula, they appear to result in a compensatory change in scapular motion.

Although the underlying mechanisms of overuse shoulder injuries have not been well established, a comprehensive review by the National Institute for Occupational Safety and Health (NIOSH) has found compelling evidence for a "positive association between highly repetitive work and shoulder musculoskeletal disorders" (Bernard, 1997). More recently Svendsen et al. have demonstrated a direct relationship between highly repetitive work activity involving arm elevation and MRI documented damage to the rotator cuff tendons (Svendsen et al., 2004). Although, the mechanism behind this relationship is not fully understood, the flow chart in Fig. 5 demonstrates one proposed model linking elevation and injury. The current study represents a novel model for the study of compromised rotator cuff function in a controlled environment.

The results of the present study are similar to the differences observed between patients with rotator cuff tears and healthy controls, where the patients with cuff tears demonstrated more upward rotation, with these differences peaking in the mid-ranges of motion (Yamaguchi et al., 2000; Paletta et al., 1997; Mell et al., 2005). However, while the largest mean increase at any humeral elevation angle for our subjects was 5.8°, patients with rotator cuff tears demonstrated much greater increases in upward rotation when compared to controls, ranging from 9° to 21° (Yamaguchi et al., 2000; Paletta et al., 1997; Mell et al., 2005). In a previous study in our laboratory, we looked at the effects of a global fatigue protocol on scapular kinematics and found similar results as in the present study: an increase in scapular rotation of approximately 6°, which peaked in the mid-range of elevation (Ebaugh et al., 2005). We used spectral shifts in surface electromyography (EMG) as an indicator of local muscle fatigue and found that the largest decrease in median power frequency was with the infraspinatus (22% drop). This was almost double the decrease of the next most fatigued muscle (posterior deltoid). It is possible that this muscle's relatively small size and function as a humeral head stabilizer accounts for this effect.

It is interesting that three separate models of rotator cuff dysfunction (nerve block, fatigue and tears) resulted in the same general pattern of increased scapular upward rotation in the mid-range of motion. Although there are clearly differences in these models, there may be some dominant underlying compensatory mechanism responsible for these changes. The larger response from the cuff tear studies is presumably due to the fact that fatigue or a nerve block does not create the same level of compromised function as a tear of a tendon. Based on the presented recovery data, a substantial reduction in muscle force was necessary to elicit an increase in upward rotation. If we assume that the force level at the first post-block measurement represented

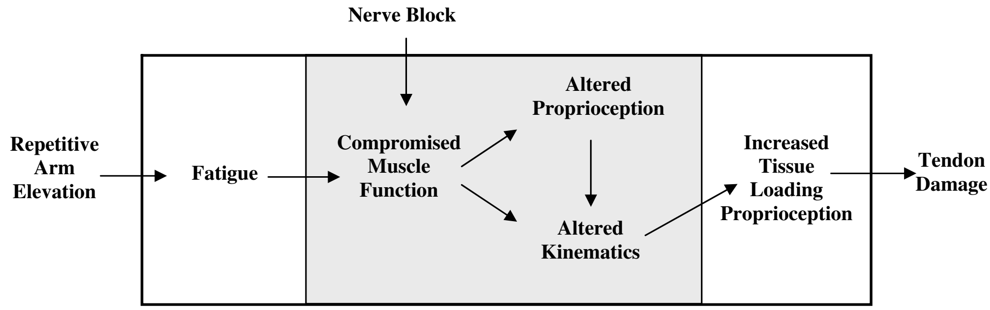

Fig. 5. Flow chart illustrating one possible mechanism for how repetitive arm elevation may lead to tendon damage.

a complete block, then with a force recovery from the block of less than 20%, no significant changes in upward rotation were observed. These results are consistent with the observations of Mell et al. (2005) who found that while rotator cuff tears resulted in increases in upward rotation, rotator cuff tendinopathy did not. However, care should be taken when comparing the results from the present study to studies of scapular kinematics in patients with impingement syndrome. While two other studies noted a decrease in upward rotation with impingement syndrome (Ludewig and Cook, 2000; Endo et al., 2001), three saw no significant differences (Lukasiewicz et al., 1999; Graichen et al., 2001; Hebert et al., 2002).

Our assessment of the extent to which a subject was blocked was based on strength measurements. We chose to measure shoulder strength during external rotation because it is a position that does a reasonable job of isolating the infraspinatus (Kuechle et al., 2000). Due to the activity of the deltoid, it would be difficult if not impossible to isolate the supraspinatus. Kuechle et al. (2000) measured moment arms around the shoulder for the same motion used to assess strength in the present study – external rotation with the arm at the side. They found that the only muscles that had a positive external rotation moment arm were the teres minor, infraspinatus, supraspinatus, middle and posterior deltoid. When they combined moment arm and cross sectional area data, they were able to estimate the contribution of each muscle to maximum external rotation torque. Based on their analysis, if the contribution of the infraspinatus and supraspinatus was removed, the resulting torque would represent 29% of the total external rotation torque generating capacity in this position. This is in excellent agreement with the data from the current study, in which nerve block resulted in a mean external rotation force level of 25% of baseline.

The changes in scapulothoracic and humeral kinematics noted in the present study may lead to detrimental conditions such as a reduction in the subacromial space, malalignment of the humeral head and glenoid fossa, or a reduction in muscle mechanics (ideal muscle length and moment arms). Alternatively, the scapulothoracic kinematic changes might be considered beneficial as they may be compensatory motions helping to maintain joint stability. The increased scapular upward rotation observed in the current study may serve to assist with elevation. With a dynamic shoulder testing apparatus, researchers at the University of Pittsburgh found that simulated tears (Parsons et al., 2002) and paralysis (Thompson et al., 1996) of the rotator cuff resulted in an inability to fully elevate the arm. However, using a similar model, Sharkey et al. (1994) reported that with simulated paralysis of the entire rotator cuff (so that the deltoid was the only active muscle), there was no reduction in maximum elevation angle. The key difference between the two models is that Sharkey et al. (1994) incorporated scapular rotations, while the studies by Parsons et al. (2002) and Thompson et al. (1996) kept the scapula fixed.

Although clinical nerve blocks are typically performed with the assistance of fluoroscopy, in order to facilitate data collection, the nerve blocks for the present study were performed in our laboratory, without any fluoroscopy. Therefore, we performed two checks to verify the integrity of the block. The first was based on the cessation of supraspinatus muscle twitch with nerve stimulation during the nerve block protocol. The second was based on the percent reduction in external rotation force from baseline, which assumes the motivation of subject is constant and there is no residual fatigue from previous trials. Although we performed the first check for all subjects, four subjects failed to meet our criteria of a 50% drop in external rotation force due to the block, with one subject demonstrating no drop in force.

The nerve block performed in the present study diminished efferent fiber information to the infraspinatus and supraspinatus muscles, which was assessed with the use of a nerve stimulator and measurements of MVC. In addition, afferent fibers innervating musculotendinous mechanoreceptors are housed within peripheral nerves innervating muscles and joints (Burke et al., 1980, 1978). Therefore, although we did not assess afferent fiber information coming from these muscles, this was presumably blocked as well. Both of these conditions would be similar to what occurs in a full thickness cuff tear. However, various branches of the suprascapular nerve also provide nerve supply to the glenohumeral joint capsule (Gardner, 1948; Solomonow et al., 1996). A loss of this afferent feedback could alter proprioceptive feedback information and the contribution of these mechanoreceptors on the conscious awareness of joint position, movement, and reflexive actions may be diminished (Safran et al., 1999; Strohm and Colachis, 1965). Therefore, it must be acknowledged that the kinematic changes observed in the post-block condition may have been due, in part, to an altered state of the afferent pathways arising from branches of the blocked suprascapular nerve, which may not be observed in patients with rotator cuff tears.

On a final note, while the loss of force generating capacity of the supraspinatus and infraspinatus in our model may be similar to the weakness observed in patients with full thickness rotator cuff tear, there are many other symptoms of this pathology that were not reproduced in this study, such as pain, limited internal rotation range of motion and crepitus (Matsen et al., 2004). Thus in reality, trying to model rotator cuff pathology with a suprascapular nerve block is probably too simplistic of an approach. However, that being said, it does provide important basic biomechanical information, in the same manner that animal (Soslowsky et al., 1996; Dejardin et al., 2001; Tillander et al., 2001) and cadavera models (Parsons et al., 2002; Halder et al., 2002; Thompson et al., 1996) do that rely on acute changes in rotator cuff function.

5. Conclusions

The results of this study, especially those for upward rotation, are in general agreement with what has been found for patients with rotator cuff tears. Although the supraspinatus and infraspinatus do not directly control the movement of the scapula, they appear to result in a compensatory change the scapulothoracic rhythm. While more work needs to be done, it appears that abnormal scapular motion patterns observed in patients with cuff tears may be compensatory in nature.

References

An, K.N., Browne, A.O., Korinek, S., Tanaka, S., Morrey, B.F., 1991. Three-dimensional kinematics of glenohumeral elevation. J. Orthop. Res. 9, 143–149.

Bernard, B.P., 1997. Musculoskeletal Disorders (MSDs) and Workplace Factors: A Critical Review of Epidemiologic Evidence for Work-related Musculoskeletal Disorders of the Neck, Upper Extremity, and Low Back—2nd Printing. National Institute for Occupational Safety and Health (NIOSH), Cincinatti, OH.

Burke, D., Hagbarth, K.E., Lofstedt, L., 1978. Muscle spindle activity in man during shortening and lengthening contractions. J. Physiol. 277, 131–142.

Burke, D., McKeon, B., Westerman, R.A., 1980. Induced changes in the thresholds for voluntary activation of human spindle endings. J. Physiol. 302, 171–181.

Cohen, R.B., Williams Jr., G.R., 1998. Impingement syndrome and rotator cuff disease as repetitive motion disorders. Clin. Orthop. 351, 95–101.

Colachis Jr., S.C., Strohm, B.R., 1971. Effect of suprascapular and axillary nerve blocks on muscle force in upper extremity. Arch. Phys. Med. Rehab. 52, 22–29.

Dejardin, L.M., Arnoczky, S.P., Ewers, B.J., Haut, R.C., Clarke, R.B., 2001. Tissue-engineered rotator cuff tendon using porcine small intestine submucosa. Histologic and mechanical evaluation in dogs. Am. J. Sports Med. 29, 175–184.

Deutsch, A., Altchek, D.W., Schwartz, E., Otis, J.C., Warren, R.F., 1996. Radiologic measurement of superior displacement of the humeral head in the impingement syndrome. J. Shoulder Elb. Surg. 5, 186–193.

Ebaugh, D.D., McClure, P.W., Karduna, A.R., 2005. Effects of shoulder muscle fatigue caused by repetitive overhead activities on scapulothoracic and glenohumeral kinematics. J. Electromyogr. Kinesiol., doi:10.1016/j.jelekin.2005.06.015.

Endo, K., Ikata, T., Katoh, S., Takeda, Y., 2001. Radiographic assessment of scapular rotational tilt in chronic shoulder impingement syndrome. J. Orthop. Sci. 6, 3–10.

Fu, F.H., Harner, C.D., Klein, A.H., 1991. Shoulder impingement syndrome: a critical review. Clin. Orthop. 269, 162–173.

Gardner, E., 1948. The innervation of the shoulder joint. Anat. Rec. 102, 1–18.

Graichen, H., Stammberger, T., Bonel, H., Wiedemann, E., Englmeier, K.H., Reiser, M., Eckstein, F., 2001. Three-dimensional analysis of shoulder girdle and supraspinatus motion patterns in patients with impingement syndrome. J. Orthop. Res. 19, 1192–1198.

Halder, A.M., O'Driscoll, S.W., Heers, G., Mura, N., Zobitz, M.E., An, K.N., Kreusch-Brinker, R., 2002. Biomechanical comparison of effects of supraspinatus tendon detachments, tendon defects, and muscle retractions. J. Bone Joint Surg. Am. 84-A, 780–785.

Harryman II, D.T., Sidles, J.A., Clark, J.M., McQuade, K.J., Gibb, T.D., Matsen III, F.A., 1990. Translation of the humeral head on the glenoid with passive glenohumeral motion. J. Bone Joint Surg. Am. 72A, 1334–1343.

Hebert, L.J., Moffet, H., McFadyen, B.J., Dionne, C.E., 2002. Scapular behavior in shoulder impingement syndrome. Arch. Phys. Med. Rehab. 83, 60–69.

Howell, S.M., Kraft, T.A., 1991. The role of the supraspinatus and infraspinatus muscles in glenohumeral kinematics of anterior shoulder instability. Clin. Orthop. 263, 128–134.

Howell, S.M., Imobersteg, M., Seger, D.H., Marone, P.J., 1986. Clarification of the role of the supraspinatus muscle in shoulder function. J. Bone Joint Surg. Am. 68A, 398–404.

Karatas, G.K., Meray, J., 2002. Suprascapular nerve block for pain relief in adhesive capsulitis: comparison of 2 different techniques. Arch. Phys. Med. Rehab. 83, 593–597.

Karduna, A.R., McClure, P.W., Michener, L.A., 2000. Scapular kinematics: effects of altering the Euler angle sequence of rotations. J. Biomech. 33, 1063–1068.

Karduna, A.R., McClure, P.W., Michener, L.A., Sennett, B., 2001. Dynamic measurements of three-dimensional scapular kinematics: a validation study. J. Biomech. Eng. 123, 184–190.

Karduna, A.R., Kerner, P.J., Lazarus, M.D., 2005. Contact forces in the subacromial space: effects of scapular orientation. J. Shoulder Elb. Surg. 14, 393–399.

Kuechle, D.K., Newman, S.R., Itoi, E., Niebur, G.L., Morrey, B.F., An, K., 2000. The relevance of the moment arm of shoulder muscles with respect to axial rotation of the glenohumeral joint in four positions. Clin. Biomech. 15, 322–329.

Kuhlman, J.R., Iannotti, J.P., Kelly, M.J., Riegler, F.X., Gevaert, M.L., Ergin, T.M., 1992. Isokinetic and isometric measurement of strength of external rotation and abduction of the shoulder. J. Bone Joint Surg. Am. 74, 1320–1333.

Ludewig, P.M., Cook, T.M., 2000. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. Phys. Ther. 80, 276–291.

Lukasiewicz, A.C., McClure, P., Michener, L., Pratt, N., Sennett, B., 1999. Comparison of 3-dimensional scapular position and orientation between subjects with and without shoulder impingement. J. Orthop. Sports Phys. Ther. 29, 574–583.

Matsen III, F.A., Arntz, C.T., 1990. Subacromial impingement. In: Rockwood, C.A., Jr., Matsen, F.A., III (Eds.), The Shoulder, vol. 2. W.B. Saunders Company, Philadelphia, pp. 623–646.

Matsen III, F.A., Titelman, R.M., Lippitt, S.B., Wirth, M., Charles Rockwood Jr., C.A., 2004. Rotator cuff. In: Rockwood, C.A., Jr., Matsen, F.A., III, Wirth, M.A., Lippitt, S. (Eds.), The Shoulder, vol. 2. Saunders, Philadelphia, pp. 795–878.

Mell, A.G., LaScalza, S., Guffey, P., Ray, J., Maciejewski, M., Carpenter, J.E., Hughes, R.E., 2005. Effect of rotator cuff pathology on shoulder rhythm. J. Shoulder Elb. Surg. 14, 58S–64S.

Nicholson, G.G., 1989. Rehabilitation of common shoulder injuries. Clin. Sports Med. 8, 633.

Paletta, G.A., Warner, J.J.P., Warren, R.F., Deutsch, A., Altchek, D.W., 1997. Shoulder kinematics with two-plane X-ray evaluation in patients with anterior instability or rotator cuff tearing. J. Shoulder Elb. Surg. 6, 516–527.

Parsons, I.M., Apreleva, M., Fu, F.H., Woo, S.L., 2002. The effect of rotator cuff tears on reaction forces at the glenohumeral joint. J. Orthop. Res. 20, 439–446.

Portney, L.G., Watkins, M.P., 2000. Foundations of Clinical Research: Application to Practice. Prentice Hall Health, Upper Saddle River, NJ.

Pratt, N.E., 1991. Clinical Musculoskeletal Anatomy. J.B. Lippincott, Philadelphia.

Safran, M.R., Allen, A.A., Lephart, S.M., Borsa, P.A., Fu, F.H., Harner, C.D., 1999. Proprioception in the posterior cruciate ligament deficient knee. Knee Surg. Sports Traumatol. Arthrosc. 7, 310–317.

Shanahan, E.M., Ahern, M., Smith, M., Wetherall, M., Bresnihan, B., FitzGerald, O., 2003. Suprascapular nerve block (using bupivacaine and methylprednisolone acetate) in chronic shoulder pain. Ann. Rheum. Dis. 62, 400–406.

Sharkey, N.A., Marder, R.A., Hanson, P.B., 1994. The entire rotator cuff contributes to elevation of the arm. J. Orthop. Res. 12, 699–708.

Sher, J.S., 1999. Anatomy, biomechanics, and pathophysiology of rotator cuff disease. In: Iannotti, J., Williams, G. (Eds.), Disorders of the Shoulder: Diagnosis and Management. Lippincott Williams and Wilkins, Philadelphia, pp. 3–29.

Solem-Bertoft, E., Thuomas, K.-Ä., Westerberg, C.-E., 1993. The influence of scapula retraction and protraction on the width of the subacromial space: an MRI study. Clin. Orthop. 296, 99–103.

Solomonow, M., Guanche, C., Wink, C., Knatt, T., Baratta, R.V., Lu, Y., 1996. Mechanoreceptors and reflex arc in the feline shoulder. J. Shoulder Elb. Surg. 5, 139–146.

Soslowsky, L.J., Carpenter, J.E., DeBano, C.M., Banerji, I., Moalli, M.R., 1996. Development of an animal model for investigations on rotator cuff disease. J. Shoulder Elb. Surg. 5, 383–392.

Soslowsky, L.J., Carpenter, J.E., Bucchieri, J.S., Flatow, E.L., 1997. Biomechanics of the rotator cuff. Orthop. Clin. North Am. 28, 17–30.

Strohm, B.R., Colachis Jr., S.C., 1965. Shoulder joint dysfunction following injury to the suprascapular nerve. J. Appl. Toxicol. 45, 106–111.

Svendsen, S.W., Gelineck, J., Mathiassen, S.E., Bonde, J.P., Frich, L.H., Stengaard-Pedersen, K., Egund, N., 2004. Work above shoulder level and degenerative alterations of the rotator cuff tendons: a magnetic resonance imaging study. Arthritis Rheum. 50, 3314–3322.

Tan, N., Agnew, N.M., Scawn, N.D., Pennefather, S.H., Chester, M., Russell, G.N., 2002. Suprascapular nerve block for ipsilateral shoulder pain after thoracotomy with thoracic epidural analgesia: a double-blind comparison of 0.5% bupivacaine and 0.9% saline. Anesth. Analg. 94, 199–202.

Thompson, W.O., Debski, R.E., Boardman, N.D., Taskiran, E., Warner, J.J.P., Fu, F.H., Woo, S.L.-Y., 1996. A biomechanical analysis of rotator cuff deficiency in a cadaver model. Am. J. Sports Med. 24, 286–292.

Tillander, B., Franzen, L.E., Nilsson, E., Norlin, R., 2001. Carrageenan-induced subacromial bursitis caused changes in the rat's rotator cuff. J. Orthop. Res. 19, 441–447.

Tsai, N.T., McClure, P.W., Karduna, A.R., 2003. Effects of muscle fatigue on 3-dimensional scapular kinematics. Arch. Phys. Med. Rehab. 84, 1000–1005.

Wong, A.S., Gallo, L., Kuhn, J.E., Carpenter, J.E., Hughes, R.E., 2003. The effect of glenoid inclination on superior humeral head migration. J. Shoulder Elb. Surg. 12, 360–364.

Wu, G., van der Helm, F.C., Veeger, H.E., Makhsous, M., Van Roy, P., Anglin, C., Nagels, J., Karduna, A.R., McQuade, K., Wang, X., Werner, F.W., Buchholz, B., 2005. ISB recommendation on definitions of joint coordinate systems of various joints for the reporting of human joint motion—Part II: shoulder, elbow, wrist and hand. J. Biomech. 38, 981–992.

Yamaguchi, K., Sher, J.S., Andersen, W.K., Garretson, R., Uribe, J.W., Hechtman, K., Neviaser, R.J., 2000. Glenohumeral motion in patients with rotator cuff tears: a comparison of asymptomatic and symptomatic shoulders. J. Shoulder Elb. Surg. 9, 6–11.